I, SCIENCE

THE SCIENCE MAGAZINE OF IMPERIAL COLLEGE

TIME

AUTUMN 2014

I,SCIENCE

THE SCIENCE MAGAZINE OF IMPERIAL COLLEGE

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I,SCIENCE, FELIX OFFICE, BEIT QUADRANGLE, PRINCE CONSORT ROAD, LONDON, SW7 2BB

EMAIL: I.SCIENCE@IMPERIAL.AC.UK

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I,SCIENCE

The concept of time is entrenched in our language and our history, it is used to mark the passing of night to day, of month to year, of decade to century. Time structures so many aspects of our lives, and yet to some it is very much an arbitrary, or even constructed, concept. It is a subject intensely studied in one form or another throughout the sciences - from the biological perception of the passing of time to the physics involved in making time travel a reality.

In this term's issue of I,Science you'll see more than just casual references to *Doctor Who* or *Einstein* quotes thrown about like there's no tomorrow. Yodit Feseha discusses biological immortality; why humans are bound by our current lifespans, and how close we might be to achieving immortal life. An account of the history of time zones from Peter Sherman explains how they were developed and why they are necessary for our modern world. Madeleine Hurry gives us a look into circadian rhythms, discussing how we have evolved an internal body clock and what impacts a disrupted sleep pattern can have. My esteemed Co-Editor-in-Chief, Iona Twaddell, has interviewed Imperial researcher, Dr Magdalena Sastre, on her work into neurodegenerative conditions associated with ageing, including Alzheimer's disease.

Of course, we have also featured some variations outside of the theme, including Andrew McMahon's explanation of epidemiological modelling and how it relates to the current Ebola outbreak in West Africa. There is also our new commentary feature, in which Stephanie Sammann and Angelia Chrysthanou battle it out over the relative benefits and disadvantages of sugars versus artificial sweeteners, and Arutyun Arutyunyan's illuminating account of Formula E: the electric alternative to traditional motor racing.

Hopefully you can find time to read it all. ■

JENNIFER TOES

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"INTELLIGENT" KNIFE ENABLES SURGEONS TO IDENTIFY CANCEROUS TISSUE DURING TUMOUR EXTRACTION

BY TIMOTHY ELLIS

A leading research group at Imperial College London has developed an 'intelligent' knife (iKnife) for use in cancer surgery. This novel technique enables surgeons to distinguish between cancerous tissue and healthy tissue in real-time during the operation.

Normally, when a primary breast cancer tumour is removed, one in five patients require further surgery to remove residual cancerous tissue. The iKnife acts to restrict the amount of healthy tissue that is removed during surgery, and make sure all cancerous tissue is extracted. Dr Zoltan Takats, the leader of iKnife research, stated that "[The iKnife] has the potential to reduce tumour recurrence rates and enable more patients to survive."

Like many surgical knives, this technology relies on the use of heat to remove sections of tissue during surgery. The innovation of the iKnife lies in its ability to detect subtle differences in cellular markers within cancerous tissue. This is realised through the vaporization of the heated tissue sample at the end of the knife. The vapour is then analysed by an interconnected mass spectrometry unit in-theatre.

Currently, surgeons are required to send biopsies to the laboratory for testing to assess the presence of cancerous biomarkers. These procedures can take up to half an hour. In many cases, these screens are carried out while the patient is still in theatre under anaesthetic, increasing the risk of surgical complications. Using the iKnife removes this requirement to send biopsies during surgery, reducing this risk of surgical complication.

Continued development of this research seeks to diversify the application of the technology to various forms of cancer. Work has already begun to create a library of biomarkers from 302 patients undergoing surgery who have been diagnosed with stomach, colon, liver, lung or brain cancer. Trials utilising this analytical method are taking place in various hospitals around London including Hammersmith, Charing Cross and St Mary's.

'iKnife' technology not only reduces costs incurred by laboratory biopsy analysis, but will also help to reduce the number of secondary surgeries and the costs associated with this. ■

Timothy Ellis is a first year PhD student studying Material Sciences

TRANSMITTING DATA USING HEAT

BY LIZZIE NORRIS

Imperial College researchers have developed a prototype technology which uses heat to transmit information over short distances. Data sent in this way has the potential to be more secure, as it is much harder to intercept compared with radio transmission, which is currently used in many wireless technologies such as car keys.

The use of a type of radiation in the infrared spectrum (blackbody) as a tool for communication has a long history of use in naval applications and is still in use today. The technology developed by the group at Imperial is the first of its kind to have potential civilian applications.

The prototype works by transmitting encoded information via a tiny incandescent light bulb which emits bursts of heat which are picked up by a receiver. The receiver detects the information in the thermal spectrum and is designed to filter out external interference. The relevant information is then decoded by a silicon chip.

Thievery of codes is a major problem of wireless door entry systems. Thieves are able to intercept wirelessly transmitted information which they then can use to break into cars and buildings. This technology makes it harder for thieves to intercept as the signal can be concealed in background noise. The system is very robust to interference and jamming as signals can only get confused if they are operating in the same spectral range, in direct line of sight and when the signalling frequency is the same.

The team have demonstrated that they can transmit information over 17cm and can transmit data at the rate of 4,000 bits per second, enabling voice and data communications to be sent. The team can partition the infrared heat into four frequency channels to transmit information and a 16 channel system is now in development. There are high hopes to increase the number of channels significantly in the future. The next step in the technology will include upgrades to the hardware so information can be transmitted at faster speeds over longer distances. ■

Lizzie Norris is a first year PhD student studying Advanced Materials

THE NEW AGE OF EPIGENETIC RESEARCH

BY CHARLOTTE MYKURA

The field of epigenetics has exploded in the last 15 years. Epigenetics is the study of the way in which DNA develops and responds to the environment; not the DNA code itself, but the way in which the DNA is folded, its chemical nature and protein content. These have huge influences over gene expression and thus cell phenotype (its observable characteristics).

A large portion of Imperial College funding now goes towards epigenetic research. Why are we seeing such rapid growth in this field?

Biological perspective has fundamentally changed. The Human Genome Project predicted that perhaps hundreds of thousands of protein-coding genes would be found nestled within our DNA. However, only around 20,000 genes were found. This is far fewer genes than are found in the humble nematode worm. How can the complex *Homo sapiens* have so few genes?

The answer lies in how our genomes are epigenetically regulated. Chemical groups and proteins bound to the DNA alter how, when and where genes are expressed or silenced. An unfathomable number of pathways alter how our DNA behaves. Epigenetic pathways are flexible and can be changed during development, making them a prime focus of research.

The institute MRC Clinical Sciences centre has a range of laboratories studying epigenetic process. For example, the Chromatin Biology Group now aims to discern mechanisms that govern epigenetic processes. Proteins known as 'Readers,' 'Writers' and 'Erasers' that modify the specific marks present on DNA regions are now being investigated.

Not only is epigenetic research essential for our understanding of how life exists, it is also furthering our understanding of diseases. Professor Richard Brown (Head of the Division of Cancer and Head of the Division of Epigenetics at the Faculty of Medicine, Imperial College) has recently published a paper in *Nature Reviews Cancer*, exploring how cancer cells can acquire resistance to drugs via alterations to their epigenetic programme.

With twelve state-of-the-art laboratories at the Clinical Sciences Centre and six epigenetic laboratories at the Institute of Developmental and Reproductive Biology, the importance of epigenetic research for medical progress cannot be underestimated, and Imperial College London is right at the forefront of this field. ■

Charlotte Mykura is a second year PhD student studying Chromatin Biology

TARGETING SEX TO STOP MALARIA IN ITS TRACKS

BY LIZ ZUCCALA

Imperial College researchers have developed a sensitive *in vitro* technique to screen drugs for their ability to block malaria transmission, making a crucial step in the effort to eradicate a disease that claims over 600,000 lives per year.

Malaria is caused by tiny single-celled parasites from the *Plasmodium* genus. As an organism with a highly complex and varied life cycle that swaps between mosquito and mammalian hosts, this pathogen presents a major challenge to scientists aiming to develop new strategies to prevent and treat infections. There is no licensed vaccine against malaria and the utility of current anti-malarial drugs is under threat from emerging parasite resistance. As such, research is focused on developing novel ways to tackle this global problem. Central to this effort is the drive to block malaria transmission and thus stop malaria in its tracks.

Malaria is transmitted from person-to-person by the bite of an infected mosquito. When a female *Anopheles* mosquito takes a blood meal from an infected person, she ingests the parasites, which undergo sexual development in her stomach. The *Plasmodium* eventually transforms into a version of the parasite that can travel through her body to the salivary glands where they wait to infect into a new host when she feeds again.

Recent research published in *Antimicrobial Agents and Chemotherapy* by Dr Michael Delves and colleagues describes a new method to test the ability of drugs to inhibit the sexual development of malaria parasites and thus block transmission. Key to their assay is its specific and high-throughput nature – they are able to screen hundreds of drugs at a time for their ability to disrupt parasite development in the mosquito midgut, without having to perform cumbersome and time-consuming experiments with mosquitos themselves. Using this technique they were able to discern crucial information about when and how drugs act on the parasite to block its function, providing a way to develop new therapies which could stop the spread of malaria. ■

Liz Zuccala is a fourth year PhD student studying Life Sciences

THE YELLOW BRICK ROAD TO IMMORTALITY

Photo: WikiMedia Commons/ Matthew Field

How would you fancy seeing the year 3000?
Yodit Feseha tells us how ageing works and if it's possible to reverse it.

The lifespan of all living organisms varies tremendously; some have lifespans measured in hours, some are measured in years and some stretch to centuries. There are even organisms that have achieved seeming immortality.

Humankind's continual reach for knowledge has allowed us to develop treatments for diseases which, in the past, would have claimed many lives. With our increasing understanding of the human body, is immortality achievable? This article explores the cause of ageing and how scientists are proceeding to further extend our life span, which could possibly lead to immortality in the future.

Flies such as the mayfly can live up to two hours, lobsters can live up to 50 years. Giant tortoises have an average life span of 100 years and the bowhead whale has an average life span of 200 years. Remarkably the naked mole rat is the longest living rodent living up to 31 years. Once *Turritopsis Dohrnii*, also known as the immortal jellyfish, has reached adulthood and reproduced, it can regenerate itself, starting its life cycle all over again.

WHAT HAPPENS AS WE AGE?

From our early 20s we already start losing our brain cells. From our late 20s the activity of the heart, lung and kidney starts to decline. Our memory, bones and muscles become weaker. Our skin loses its elasticity, becoming wrinkly. The immune system is weaker and becomes more susceptible to infection. Reduction in the pigment melanin results in grey and eventually white hair.

FREE RADICAL THEORY OF AGEING

One theory of ageing is that free radicals decrease the lifespan of an organism by influencing ageing associated diseases such as cancer, stroke, diabetes and many more. A free radical is an atom, molecule or ion that has unpaired electrons causing it to be highly reactive towards other substances and itself. As we age, free radicals accumulate in our bodies as the result of several factors such as the breakup of large molecules, ionising radiation, heat and chemical reactions. On a molecular level, the human body therefore accumulates free radical damage to the cell as it ages.

Accumulation of free radicals has been implicated as the cause of DNA, protein and lipid (fat) damage. This damage can lead to mutations in the genome causing various diseases and cell death.

In contrast, free radicals can also assist immune cells such as macrophages in fighting infection and are also associated with the cell communication process.

MITOCHONDRIAL THEORY OF AGEING

An older individual has a higher amount of damaged/mutated DNA, oxidised proteins, and lipids (which are essential for energy storage, cell signalling and structural components of the cell membrane). The mitochondria (energy producers in cells) in particular are more susceptible to this type of damage as they are more exposed to it. They also have a less efficient DNA repair mechanism compared to nucleic DNA. A cell can contain hundreds of mitochondria which are involved in energy production by synthesising Adenosine triphosphate (ATP); a high energy molecule that stores the energy we use.

Oxidative substances, by-products of the ATP synthesising process, are released into the mitochondria and the cell. Those oxidative substances, which can include non-free radicals can cause damage to the cell, including the essential enzymes. As a result, they can damage the function of the mitochondria, leading to a low level of ATP production.

This is one of the main causes of ageing, as the cell cannot function well with insufficient energy. Thus, oxidative substances lead to age related diseases causing a shorter lifespan. Interestingly the longest living mammal, the naked mole rat has the ability to slow their metabolism, thus preventing oxidative damage in cells.

Reducing the number of calories eaten in mice and in humans has been shown to result in a decline of oxidative substances and therefore less damage to cells. This is because a high calorie diet can increase the level of free radicals

Turritopsis Dohrnii, the immortal jellyfish

produced in the mitochondria from metabolism. Therefore it is evident that a healthier lifestyle, with a low calorie diet and plenty of exercise can lead to a longer life.

THE AGLETS OF OUR CHROMOSOMES

Telomeres are often compared to the plastic tips of shoelaces (aglets). They are a repeated DNA base sequence found in the tip of chromosomes which prevent chromosomes from fusing their tip ends with each other. During every cell division the telomeres lose 25-200 bases of their sequence. Once this reaches a certain length, the cell can no longer replicate and undergoes programmed cell death.

These telomeres are regulated by an enzyme known as telomerase. This enzyme can elongate the telomere's sequence and is highly active during developmental stages. The absence of telomerase in adult cells, leads to an ageing body.

So why don't we just inject ourselves with telomerase? Well, telomerase is associated with cancer cells. HeLa cells (an 'immortal' human cell line isolated in cervical cancer patient, Henrietta Lacks) are used often in experimental biology, and do not suffer any form of cell death due to the highly active presence of telomerase.

THE REVERSIBLE PROCESS OF AGEING?

Theories of ageing explain its process, but it has been hard to pin down how it can be reversed. Consumption of anti-oxidants, for example, may reduce oxidative substances in the cell but excessive amounts can also cause harm to the body. However, Professor David Sinclair and his research team at Harvard University have investigated an ageing process that is reversible.

The nucleus encodes genetic material that is useful for the mitochondria, which in return generate energy the nucleus requires.

> **SO WHY DON'T WE JUST INJECT OURSELVES WITH TELOMERASE? WELL, TELOMERASE IS ASSOCIATED WITH CANCER CELLS**

This communication between the two parts is essential for a healthy functioning cell. In the bodies of younger people, there is very good communication between these elements but this gets weaker as the cell ages. This is caused by a low level of nicotinamide adenine dinucleotide (NAD+) in the cell. This causes certain enzymes dependent on NAD+ involved in the interaction between mitochondria and the nucleus to not function properly.

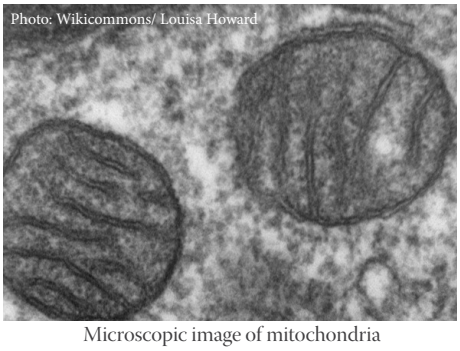


Photo: Wikicommons/ Louisa Howard

Microscopic image of mitochondria

For example SIRT1 (NAD+ dependent deacetylase) is an enzyme important for this communication and it has been observed at a low level in older laboratory mice as the result of low NAD+ level.

Activating SIRT1 artificially in mice extended their life span significantly. The same mice who saw their life span extended in this way also experienced an improved metabolism and less physiological and cognitive damage. In addition, fewer age related metabolic diseases were experienced later in life and mice were generally healthier. The artificial activation of SIRT1 also resulted in regeneration and restoration of the heart muscles, whilst mitochondrial activity was restored. A drug has been developed to do exactly this in humans, which currently is undergoing clinical trials.

If the outcome of this is similar to that seen in mice, extending human lifespan might be possible in just a few years. Our understanding of many biological processes has advanced enormously in the last ten years, in particular because advances in technology have changed how we can analyse biological data. So why can't immortality be achievable in the future? ■

Yodit Feseha is studying for an MSc in Human Molecular Genetics

TIME FOR A RETHINK?

Jai Dongre explains why time flies when you're having fun and the psychological basis of other temporal illusions.

Time. Physicists define it as the fourth dimension. Philosophers claim it as a conception of the mind. Mathematicians simply know it as *t*.

Our perception of time has been a significant field of study for psychologists and neurologists alike. Leaving behind the relativistic effects of time dilation, our perception of time in daily life is subject to many factors. Everyone has been in a lecture where time doesn't seem to pass: the feeling that one of Homer's great epics could have been recited in what the clock shows to be a couple of minutes. Numerous studies have found that boredom, negative emotions and a lack of motivation increase the perceived duration of time for a certain task. These are called temporal illusions. Just like optical illusions alter what we see from what is really there, temporal illusions alter our idea of how much time has passed.

> "TEMPORAL ILLUSIONS ARE SO COMMONPLACE THAT WE CEASE TO QUESTION THEIR ABSOLUTE BIZARRENESS"

Temporal illusions are so commonplace that we cease to question their absolute bizarreness. None of our traditional five senses accounts for the perception of time. We do not see time pass, or hear or smell it go by. Our 'sense' of time is a crucial part of our brain's perception of reality, yet we barely notice when it is simply wrong.

For example, time seems to pass faster with age. Even though there are no definitive explanations for this, the theories that have been proposed are quite compelling. One hypothesis is that any given time period for an adult, constitutes a smaller fraction of their life than for a child and so it may seem shorter to them. Another theory, relevant to university life, uses stress as the explanation. Most students know the feeling of not having enough time to do things. Studies by Wittman and Lehnhoff from the University of Munich have shown that people often reinterpret this stress as the feeling that time is passing faster.

Other temporal illusions appear in places you would not expect. A study by Wearden, Todd and Jones at Manchester University found that visual stimuli appeared shorter in duration than auditory stimuli of the same length. It seems that time not only plays a part in the perception of our senses, its effects are also variable.

At this point, we must pause to consider whether we could ever have a perfect, or even close to perfect, sense of time. Is it realistic to envisage a day where clocks are not necessary and you always know the precise time? Efforts to improve our senses are well underway. Sight and hearing already have a variety of aids available and touch restoration is already becoming a reality. The future could well hold devices or

Photo: Aaron Hall

techniques for perfecting our taste and smell, but could our sense of time ever be fixed if we still haven't understood how it works?

For the moment, at least, it seems that the effects of temporal illusions are here to stay. Our sense of time may be analogous to our sense of direction, and just like GPS technology will often prove our intuition wrong, we may have to continue looking at a clock for an accurate reading of the time. Any way around this can only come in the distant future, after we've improved our understanding of how the human brain functions.

Even though we may never see the day when boring family dinners fly by and interesting conversations seem to last forever, our brain's perception and processing of time is truly fascinating. We should stand in awe, for time is probably the single most essential aspect of our reality, and without it, the world as we know it could never exist. ■

Jai Dongre is a second year undergraduate studying Biomedical Engineering

THE WORLD'S GOT RHYTHM, CIRCADIAN RHYTHM

The Latin words *circa diem* translate to 'about a day'. Speaking of old things, circadian rhythms are pretty ancient. They are a result of life that evolved on a planet that rotates over a period of 24 hours. Being able to anticipate daily environmental fluctuations is remarkably useful, hence these biological clocks are virtually ubiquitous. The clock components are fairly basic: simply a handful of proteins and some ATP (the cellular energy transfer molecule) can suffice. But disrupting them causes complications ranging from jet-lag to cancer. Circadian rhythms are therefore fascinating on a molecular, medical and evolutionary level.

THE DISCOVERY OF CIRCADIAN RHYTHMS

Biology is dynamic; even seemingly motionless plants change during the course of the day. Linnaeus, the father of taxonomy, demonstrated this in 1751 when he designed his 'flower clock'. If different plants with variations in the opening and closing times of their flower buds were planted in a circle, they could theoretically tell you the time!

But it wasn't until the 1970s and the genetic revolution that this research really took off.

The first circadian genes were discovered in fruit flies by Seymour Benzer and his student Ron Konopka. They controlled when flies emerged from their pupal cases and were named 'Period'.

In 1997 a second gene was identified in mice, aptly named 'Clock'. Clock mutants exhibit variability in sleep-wake cycles. A wild-type mouse kept in a dark cage has a circadian cycle slightly shorter than 24 hours. Animals with one copy of the mutated Clock gene have abnormally long cycles and mutants with two abnormal copies of the Clock gene have asynchronous cycles, even in normal light-dark environments.

> "YOU ARE PROBABLY LESS SAFE DRIVING BETWEEN 4AM AND 6AM THAN WHEN YOU ARE LEGALLY DRUNK"

HOW DO YOU BUILD A CIRCADIAN CLOCK?

The basic components of the body's biological clock vary across the tree of life. Whilst the proteins used to build the clock vary, the logic of the architecture is conserved. Fundamentally, it requires an oscillatory mechanism that follows a 24-hour period.

The simplest biological oscillator exists in the cyanobacterium *Synechococcus elongates*, the core of which involves only three proteins: KaiA, KaiB and KaiC. Here, KaiC becomes increasingly phosphorylated then dephosphorylated over a period of 24 hours. These proteins will even continue to function as a basic circadian clock *in vitro* in the presence of ATP and magnesium.

In mammals, the core oscillator unit of circadian rhythm is slightly more complex. The transcription factors (proteins which help gene expression) CLOCK and BMAL pair up and promote transcription of the genes Per and Cry. PER and CRY proteins join together and travel back to the nucleus to suppress the transcription of Clock and Bmal. This happens slowly, producing the 24-hour cycle. Other genes governed by circadian rhythms are activated or suppressed as the levels of CLOCK and BMAL rise and fall. This may be noticeable, like the secretion of a hormone telling you it's time to wake up, or go completely undetected, such as increased DNA repair in the late afternoon.

This oscillatory mechanism can free-run by itself, but cell-cell signalling is required to synchronise the rhythm with external environmental cues, such as light.

CIRCADIAN RHYTHMS AND THE BRAIN

Let's take a giant evolutionary leap from single-celled prokaryotes to mammals and discuss how our circadian rhythm is organised anatomically.

When we see things, light enters the visual pathway through our pupils. This signal is transduced by photoreceptors in rod and cone cells, causing retinal neurons to fire, which finally forms an image interpreted by our brain. But another light pathway exists, one that entrains your biological clock. This time the light signal is picked up by special cells known as intrinsically photosensitive Retinal Ganglion Cells (ipRGCs), which contain the photopigment melanopsin.

The discovery of ipRGCs was made by Professor Russell Foster, former Chair of Molecular Neurosciences at Imperial, and his team. This was a contested but momentous discovery, as it was previously thought that no photosensitive cells other than rods and cones

Circadian rhythms run the biological world like clockwork from the inside, allowing organisms to anticipate environmental changes over the course of a day. Madeleine Hurry tells us how they work, from the genes in a bacterium to the human brain.

Photo: Flickr/ Pedro Ribeiro Simões

existed. The ipRGCs stimulate neurons in the suprachiasmic nucleus (SCN) in the brain. The SCN is a tiny structure within the hypothalamus. But it is the master regulator for circadian rhythm because it synchronises the rest of the cells in the body with the 24-hour light-dark cycle.

Interestingly, some non-mammalian vertebrates, such as frogs and lizards, possess a light-sensitive organ found on the tops of the heads. This is called a parietal eye, also known as the 'third eye.' In mammals this organ is absent, but the analogous structure is the pineal gland, which produces melatonin, the hormone responsible for sleep patterns.

WHAT IMPLICATIONS DO CIRCADIAN RHYTHMS HAVE?

Perhaps you just can't seem to get to sleep before midnight. Meanwhile your friend can't sleep past 7.30am, even on weekends. The reason is, of course, your personal circadian rhythm.

Whilst healthy people can generally adapt their routine when required, those who aren't may have a circadian rhythm sleep disorder (CRSD), so cannot adapt their cycle to conventional social hours. This causes disrupted or inadequate sleep, which can lead to depression, weight gain, and various other diseases. CRSDs can be caused extrinsically, by shift work or jet lag, or intrinsically, by a mutation in a circadian gene.

You can feel the effects of an asynchronous circadian rhythm when you suffer from jet lag. A common plague of the transmeridian traveller, jet lag is caused by an abrupt change in time zones. This means your circadian rhythm is out of sync with your new location, causing sleep disturbance, irritability, headaches and digestive problems.

Both physical and cognitive performance is paired with circadian rhythm. For example, you can swim three seconds faster at 8pm than 6am simply due to the balance of circadian-controlled hormones in your body. Similarly but more seriously, melatonin and other sleep hormones which are released at night reduce your cognitive ability, so much so that you are probably less safe driving between the hours of 4am and 6am than when you are legally drunk!

Metabolism and circadian rhythm are also interlinked, so lack of sleep causes hormonal imbalances. Leptin, responsible for fullness, and ghrelin, which stimulates hunger, exist in equilibrium. But sleep disruption will tip the equilibrium towards ghrelin and increase its production by 28%. In real terms, this means that people who get less than five hours sleep per night have a 50% likelihood of being obese! Also related to obesity, the circadian clock gene BMAL1 has been associated with type 2 diabetes and hypertension.

Many genes involved in regulating circadian rhythm have been implicated in psychological diseases. For example, SNAP25, which is associated with schizophrenia in humans, causes disrupted sleep cycles and hormone release when mutated in mice. Similarly, ADHD and sleep disorders are often found together, although it is not yet known if there is a causal relationship between the two.

Circadian rhythms also need to be considered in pharmacology. The efficiency of nucleotide excision repair (a form of DNA repair) peaks in the late afternoon, after most UV-related DNA damage has accumulated. The synthesis of the proteins required for DNA repair are controlled by the circadian clock. Therefore, timing the administration of chemotherapy to coincide with this peak could increase the efficacy of the drug.

Conversely, studies have also shown that certain drugs can be more toxic at different times of day. If a group of mice are given cyclophosphamide (a chemotherapeutic agent) at dusk, there is 20% mortality. Give them the drug at dawn and all of them will die.

Ultimately, circadian rhythms alter your body's biochemistry causing profound effects. Next time you feel like falling asleep in your 9am lecture, consider that it is due to the action of some evolutionarily ancient genes! ■

Madeleine Hurry is studying for an MRes in Experimental Neuroscience

A TIMELINE OF TIMEKEEPERS

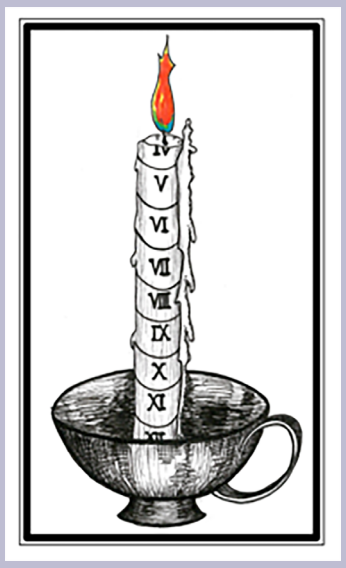
Keeping time: **Kruti Shrotri** *looks at the different timekeepers our civilisation has used over the last five thousand years.*

SUNDIAL

The sundial is the earliest known timekeeping device. The first known sundial dates to over five thousand years ago, and consisted of only a vertical stick. The shadow-casting part of a sundial is known as the gnomon; in traditional sundials, the gnomon tends to be a triangle, such that its hypotenuse is parallel to the Earth's axis.

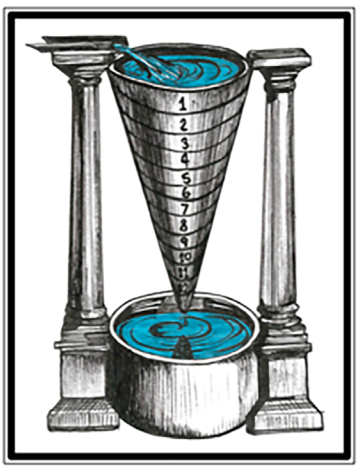
CANDLE CLOCK

The earliest mention of the candle clock dates back to the 6th century, but it is usually attributed to Alfred the Great who lived a few centuries later. His candle clock comprised of six candles, each twelve inches high. One inch of the candle would burn in approximately 20 minutes such that six candles would burn in 24 hours.



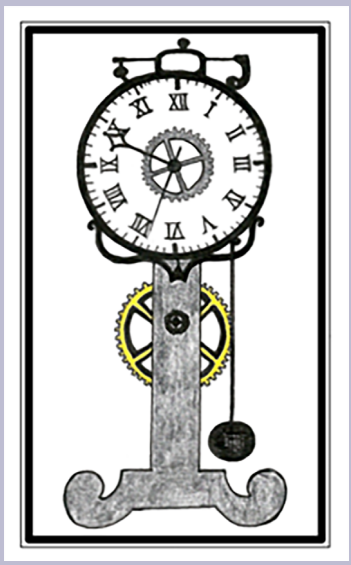
CLEPSYDRA

A clepsydra measures time through the gradual flow of liquid. The oldest specimens found were Egyptian, dating back to the 14th century BC. These used water. However, since water freezes at 0°C, later versions used mercury instead, which freezes at a much lower temperature of -38°C. Galileo used a mercury clepsydra in as late as the 16th century in his experiments with falling bodies.



MECHANICAL CLOCK

Mechanical clocks have been used since the 8th century. They tended to be driven by water, and later by weights. Soon after 1600, Galileo discovered that the motion of a pendulum could be used to regulate clocks, but it wasn't until 1656 that Chistiaan Huygens built the first pendulum clock. These increased timekeeping accuracy from around 15 minutes per day to 15 seconds per day.



Illustrations by Rosie Woodcock

POCKET WATCH

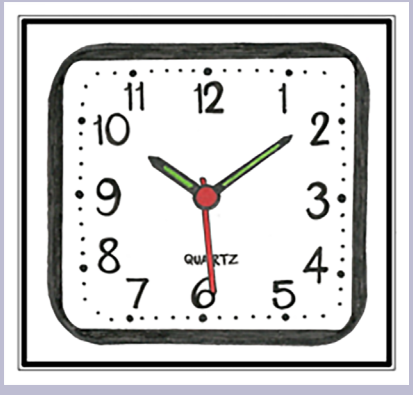
The first pocket watch, invented around the 15th century, had an accuracy of only several hours per day. However, in the 17th century, Robert Hooke and Christiaan Huygens developed the balance spring. The balance spring, which may be thought of as a watch's equivalent of a clock's pendulum, decreased inaccuracy from several hours to just ten minutes per day.

ATOMIC CLOCK

The first atomic clock was designed by Louis Essen and built in 1955. These clocks keep time using the properties of a caesium atom and are accurate to one second every few hundred million years. The invention of the atomic clock resulted in a new time standard, one that we still use today. According to this time standard, one second is equal to 9,192,631,770 cycles of a light wave emitted by a caesium atom.

QUARTZ CLOCK

The first quartz clock was built in 1927. These clocks use a quartz crystal, made from silicon dioxide, to keep time. An electric current, from a battery, makes the quartz crystal oscillate at 32,768 times per second. These oscillations regulate the gears that make the clock tick. They are accurate to one second per two days.



LOOKING FORWARD

Scientists have succeeded in building quantum clocks, which keep time by counting the vibrations of an electrically charged aluminium atom. This atom vibrates at 1.1 quadrillion times per second, meaning the clock is accurate to one second per few billion years. The quantum clock offers the potential for a new time standard, more precise than ever before. However its design is complicated and needs further development before it can be considered a serious contender.

Kruti Shrotri is studying for an MSc in Science Communication

TIME: GOING PLACES

What's the truth about the relationship between space and time? Isobel Nicholson tells us...

Space is a very familiar concept. We intuitively understand distance – when we go for a walk, we cut the corner without ever needing Pythagoras's theorem. If you want to get to the shop across the square, you could take three steps forwards and then four to the left, but you will probably just walk five steps in a straight line. It's easy to prove, even if you include the third dimension (up and down): the square of the distance travelled by the direct route is the sum of the squares of what we call the three orthogonal directions (x, y and z).

The fourth dimension is far less intuitive. We know that time is a dimension because it fulfils all the criteria: we have been measuring 'distances' in time for thousands of years and you only need to look at the recent cinema releases to see that we are comfortable with the concept of going 'forwards' and 'backwards' in time! We also instinctively understand that time acts in a fourth, very different 'direction' which is independent of the spatial directions.

However, time is far more beautiful and complicated than simply an addition to our spatial comfort zone. The key difference is not the physical difference between being able to see for ourselves that space exists in front of us while simply having to assume that the future will be there. This difference is strange, but it is not a deep question of philosophy related to the way we experience reality: after all, we do not 'see' anything but simply interpret a complex series of mid-range electromagenetic signals. The key difference is that if time was like one of our three spatial dimensions, we would be able to have a straight line through space, where the value of time didn't change, just as we can have a straight line through time where your position doesn't change. But you can't stand still in time, or move backwards, you can only go forwards.

> "THE FASTER YOU TRAVEL THROUGH SPACE, THE SLOWER YOU EXPERIENCE TIME... IT IS SIMPLY THE WAY OUR UNIVERSE WORKS"

But do we all move forwards in time at the same pace? It turns out that the faster you travel through space, the slower you experience time. This is not a question of biology or consciousness – a clock that has been left on an aircraft will show a fractionally earlier time than one on the ground. It is simply the way our universe works, and the only reason that we don't understand it intuitively is because we don't travel fast enough. As you travel faster and faster, time slows down until at some point it is very, very close to stopped. This speed has been experimentally found to be 670,616,629 miles per hour, the speed of light. Only particles with no mass, like photons, can travel at this speed and nothing can break this barrier.

According to special relativity, we can quantify the relationship between time and space by trying to think of some kind of Pythagoras's theorem that will measure the total 'distance' travelled through both space and time at once. We know that the square of the distance between two events in three-dimensional space just adds up the squares of orthogonal directions. But as we have seen, when you travel quickly, though you travel further in space, you travel slower in time, which we need to take into account in our equation. So the square of the distance between two events in spacetime adds up to the square of the orthogonal spatial directions minus a quantity proportional to the square of the duration in time. This 'distance' in spacetime is called the 'spacetime interval.'

The spacetime interval has a special property. Take the following scenario. I am on a train moving at a constant speed, sitting in a seat, watching the ticket officer walk down the aisle. I see him move a few metres. However, if I am instead on the side of the train track, and watch the ticket officer walk down the aisle, I see him fly by, moving many metres. This because the train, as well as the ticket officer, is moving relative to me. You can see that the distance someone else moves in three-dimensional space changes according to how fast you yourself are travelling. However, in four-dimensional space, the spacetime interval of two events always stays the same, no matter how fast you yourself are travelling. So if I measured the spacetime interval of the ticket officer moving when sitting on the train, it would be the same as if I measured the spacetime interval of the ticket officer moving when sitting on the side of the train track. The fact that spacetime intervals don't change is called 'Lorentz invariance' and the spacetime interval itself is called an 'invariant.'

Things that don't change, like invariants, are often called symmetries, and they have an important mathematical property, discovered

by Emmy Noether. This is that every symmetry has a conserved quantity associated with it. For example, if distance doesn't change in a particular physical system, then momentum is conserved; if time doesn't change, then energy is conserved. The symmetry of Lorentz invariance means that in spacetime, the energy and momentum together are conserved: Time invariance is therefore intricately tied up with the conservation of energy.

Unfortunately, spacetime is all a bit more complicated that than. The mass of an object actually bends spacetime, making other masses travelling towards it appear to accelerate. We call this phenomenon gravity. This idea is often modelled by a ball on a rubber sheet – the ball's mass produces a dip in the rubber sheet, so that any mass on the edge of the rubber sheet would accelerate towards to the middle. Taking these sorts of accelerations into account moves us away from the theory of special relativity to the theory of general relativity.

But we've seen at least one thing: time's role as a dimension is essential. So many factors, from the finite speed limit of the universe to the conservation of energy itself, depend on time and its relationship with space. It is the interplay between space and time that makes it possible for the universe as we know it to exist ■

Isobel Nicholson is studying for an MSc in Quantum Fields and Fundamental Forces

Photo: Flickr/ Xynn Tii

SCIENCE BEHIND THE PHOTO

Photo and words by Jessica Norris

This isn't just a line across a courtyard. This is the prime meridian line, and the home of Greenwich Mean Time (GMT), the world's timekeeping centre.

This imaginary line, running from North to South Pole with longitude 0°, marks the start of every new day. From here the earth divides into east and west, just as the equator denotes north and south. It not only enables us to navigate, but has kept the world's clocks ticking to the same 24 hour clock for over 125 years. Though, new technology threatens to redefine time.

GMT was first proposed by Royal Astronomers to equate the sun movements to local time. Later, GMT found its way into everyday use following the need for a standardised time, due to the rise of the railway. After gaining worldwide approval in 1884, time zones around the world have been measured in relation to a reference time generated from this line.

Since 1960, atomic clocks have taken over. Using the reaction of caesium atoms, these clocks can measure the length of every second with extreme accuracy. Since then we have also realised that the Earth's rotation slows two thousandths of a second every day. This means atomic time and Earth time, may slowly drift apart; so leap seconds have been added to keep them in sync. As such, a compromised version of GMT, Co-ordinated Universal Time, UCT, controls time.

French scientists have recently suggested a new system based entirely on atomic clocks without reference to Earth's rotations. This would eliminate the need for leap seconds, but would also cut long held ties between the rising of the sun and time, rendering GMT obsolete. While their proposition has yet to come to fruition, it reminds us that the Greenwich Meridian line unlike the equator is an arbitrary concept. It is not determined by nature, but is proposed by science and debated by governments. ■

Jessica Norris is studying for an MSc in Science Communication

INTERVIEW WITH DR MAGDALENA SASTRE

Dr Magdalena Sastre is a senior lecturer at Imperial College London in the division of Brain Sciences. Her research forcuses on the molecular mechanisms behind Alzheimer's disease. In this Time issue of *I, Science*, we wanted to ask her how time can affect our brains, in terms of the consequences of age-related disease and how we can try to treat or prevent neurodegenerative disease.

So, what happens to our brains when we age?

When we age, we lose neurons and the brain usually shrinks. This is in a normal ageing population. But in cases of Alzheimer's disease this atrophy in the brain is more pronounced, and people lose neurons in certain areas of the brain that are involved in learning, reasoning and in orientation like the hippocampus or the cortex.

Can you tell me about some of the molecular mechanisms behind Alzheimer's disease?

In the brain of an Alzheimer's patient, we have the deposition of certain proteins. One is called amyloid beta protein that makes amyloid plaques, and other is phosphorylated tau that aggregates inside the neurons and the neurons tend to die. In the brain of these patients, surrounding these [amyloid] plaques there are glial cells – support cells that can secrete inflammatory mediators. These inflammatory mediators could mediate neuronal death, and many people believe that this is the way that amyloid is toxic for the neurons and responsible for the neurodegeneration that occurs in the brains of these patients.

What kind of research are you doing at the moment?

We are doing two kinds of research. One is *in vitro* research, using cell lines, and we are trying to investigate the mechanisms and pathways by which inflammation affects the formation of amyloid and neuronal death. And on the other hand, we are using animal models in which we are trying different therapies to see which one could be effective for treating the disease. And many of these therapies are anti-inflammatory therapies.

Any promising results?

Yes, our last paper was on a growth factor called FGF2, or fibroblast growth factor, and we have seen that the animals treated with this growth factor, have improved memory and reduced pathology.

How much can environmental factors influence the development of Alzheimer's disease?

There are certain environmental factors, like brain trauma or type-2 diabetes that could affect the increase of inflammation, so we are trying to find out if these could affect the progression of the disease.

What is the best way to prevent Alzheimer's disease?

There are lifestyle factors that are very important, like doing exercise and eating the right food. Smoking is also dangerous for the brain, as is drinking alcohol. The most protective factor for Alzheimer's disease is physical exercise: it's not only good for your heart, it's also good for your brain!

You can do the basic research, but policy is obviously needed to implement it effectively. What can policymakers do to help reduce Alzheimer's disease?

There are two things they can do, they can give more money for research, not only for translational research to find the medicines to cure Alzheimer's, but also to investigate the cause of Alzheimer's. That is

Photo: Charlie Corbett

Iona Twaddell spends some time with Dr Magdalena Sastre to learn more about age-related diseases.

what we are doing in my lab, basic science research. And the other thing is to try to get people to improve their lifestyle and make people aware that when they have some symptoms of dementia they should go to the neurologist or the psychiatrist because the sooner they get diagnosed the better.

How important is early diagnosis?

They are investing a lot of money in early diagnosis because unfortunately all the clinical trials that have been done so far have been done in severely ill Alzheimer's patients, when they have lost a lot of neurons. And the neurons do not regenerate. So if you can stop the progression of the disease at earlier stages with an early diagnosis of the disease, for instance using imaging, or with analysis of biomarkers in the blood, or the CSF [cerebrospinal fluid], then people could get treatment earlier, to have more chance of not getting worse.

Why is it so important to research these diseases?

There are several reasons. One is to know which is the cause of the disease. If we know the cause of the disease or what provokes Alzheimer's, it will be much easier to find a treatment for the disease. Unfortunately Alzheimer's, Parkinson's and other neurological diseases don't have a cure. They just have some treatments that ameliorate the symptoms but they don't cure the disease, so we need to find better treatments. And secondly because all these diseases are caused when people get older. And now the population in the world is ageing, so it's very normal to find people that are 80, 90 years of age, and the probability of getting Alzheimer's at this age is almost 40% so we need to stop this because it generates an enormous cost.

How important is the inter-disciplinary approach of getting clinicians and researchers working together?

It's very important because you have to work closely with the patients to see if your results are reflected in an Alzheimer's patient. It's very important if we have a treatment that we have shown that works in animals and in cells that it's also working in humans, because we are very different. So we need to interact with the clinicians and organise clinical trials with them.

What is the most important question in neuroscience?

In my field of research I think the most important question is to see how we can stop neurodegeneration because so far nobody has managed to do that and it affects not only Alzheimer's disease (my field) but other neurological diseases like multiple sclerosis, Parkinson's disease or ALS [amyotrophic lateral sclerosis]. So I think if we try to find out why the neurons degenerate and how to stop that, that will be the future for all these kinds of diseases.

What is your favourite part of your work?

I like working in the lab because even though I am a lecturer, and I'm supposed to be in my office writing grants, I also like doing experiments – it's like cooking – and it's very exciting when you have a very good result and you think "we are going to publish this in a very good journal!"■

Iona Twaddell is studying for an MSc in Science Communication

Watch a video of this interview on our website, www.isciencemag.co.uk

MAKING TIME

Why does Russia have 11 time zones while China has just one? Peter Sherman investigates the history, politics and technology behind some of the world's most incongruous ways of telling time.

Humankind was once dependent on the location of the sun to determine the time of day. With the onset of rail transport and the telegraph, however, solar time quickly became obsolete. As people began travelling great distances in short periods of time, it became necessary to standardise local time scales.

Greenwich Mean Time (GMT) was initially established in 1675 for mariners at sea, but it took centuries to catch on as the standard time zone. Nowadays the world is partitioned into 24 standard time zones, which correspond to the 24 hours in each day. The time at any location is determined by its longitudinal proximity to Greenwich, London – every 15˚ in longitude alters the time by one hour.

Despite this attempt at standardisation, for several historical and technological reasons time zones still come with their fair share of bizarrely interesting quirks.

Although India spans nearly three time zones, it observes only one uniform time zone, Indian Standard Time (IST = GMT+5.30), across the entire country. IST was originally created to simplify railway times countrywide in the 19th century. Due to the inefficiencies of having one time zone for a nation that covers nearly 3,000km longitudinally, eastern regions, like Assam, demand timezone reform.

While regions in India are looking to get ahead of the times (pun intended), Russia appears to be taking steps in the opposite direction. In early 2010 Vladimir Putin signed three decrees to abolish two of Russia's 11 time zones in an attempt to unite other regions and increase business relations with Moscow. Putin may have taken this idea from China.

China Standard Time (CST = GMT+8.00) is used throughout all of China. From 1912 to 1949 China was divided into five separate time zones which, geographically speaking, made sense. Once the People's Republic of China was established, however, the government abandoned all of the time zones, and sought to establish a single time zone based on Beijing time for the entire nation. This is especially problematic for cities in western China, such as Kashgar, where the sun typically sets at midnight CST.

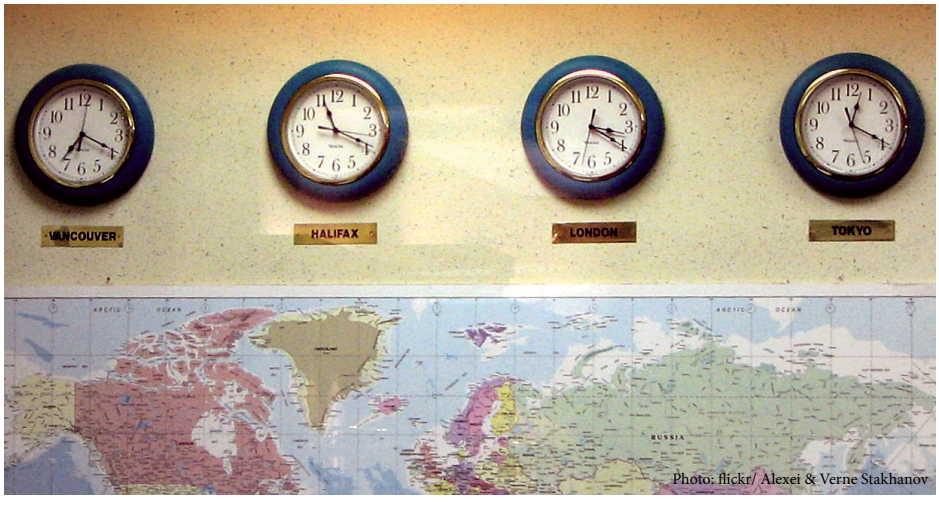


Photo: flickr/ Alexei & Verne Stakhanov

In another example of time zone weirdness, Spain is an hour ahead of the UK despite the fact that the two nations are in the same 15˚ longitudinal gap. The reason for this discrepancy dates back to over seven decades ago, during World War II, when Spanish dictator Francisco Franco turned the clocks ahead an hour to match with their ally, Nazi Germany. Even though their time zone now seems outdated and nonsensical, Spain has yet to change it back to what it once was.

Well-known by most of Western civilisation is the infamous Daylight Savings Time (DST). The main purpose of DST is to start the day earlier, to correspond with the earlier rise of the sun in the spring and summer, which gives us more time awake in daylight. The process can be difficult to track as there are more countries that have not adopted DST than those that have. It is especially confusing in North America where there are regions of Canada, the US, and Mexico that have forgone DST while the rest of their nations have stayed put.

Strangest of all is the International Date Line, an imaginary line running through the Pacific Ocean that marks a difference of one calendar day depending on which side of the line you're on. Travelling from west to east over the IDL moves you ahead one full day, and vice versa for east to west.

While time zones can be bring about some confusion, they are a necessary evil for a world that is more interconnected than ever before. ■

Peter Sherman is a second year undergraduate studying Physics

THE LIVING DEAD

Benno Simmons discusses extinction debt and why biodiversity might be in an even worse state than we realise.

Global biodiversity is declining at an alarming rate as extinction drivers, such as habitat loss, invasive species and climate change, cause huge and immediate biodiversity loss. These are the statistics we see reported in newspapers, magazines and the journal articles every day and they don't paint a pretty picture.

However, things might be worse than we realise. While many species become extinct soon after an environmental perturbation, others take a significant amount of time to disappear: a habitat could be destroyed, but a species living within it not become extinct for another fifty years. This is the worrying phenomenon known as extinction debt, the time lag between an environmental disturbance affecting a species, and its final disappearance. If it's widespread, there could be large numbers of extant species, committed to extinction, 'living dead' species simply waiting to disappear unless something is done.

This may sound worrying but it's important to remember that our understanding and knowledge of extinction debt is actually quite limited. The number of national or regional scale studies is small, so we don't really know how widespread the problem is, though preliminary evidence is concerning. For example, a recent study from Dr Rob Ewers' group at Imperial found that, in the Brazilian Amazon, while local extinctions of forest-dependent vertebrate species have been minimal, more than 80% of extinctions are still to come, incurred from historical habitat loss. Another study found that, in Africa, most countries have a debt equal to 30% of forest primate fauna.

We also don't know much about how different species might be affected. So far, studies have generally focused on vascular and forest cryptograms (an assembly of organisms like lichen and algae), with studies of animals, other than birds, being in the minority. Theoretically, however, species with low turnover rates (such as perennial plans or mammals) and those with habitat specialisms are the most likely to incur a debt. There's also more likely to be a debt in landscapes where habitat loss has occurred quickly.

Photo: flickr/ Ray Morris

Ultimately though, we don't know much, and there is an urgent need for extinction debt research concentrating on greater range of organisms and conducted at larger scales.

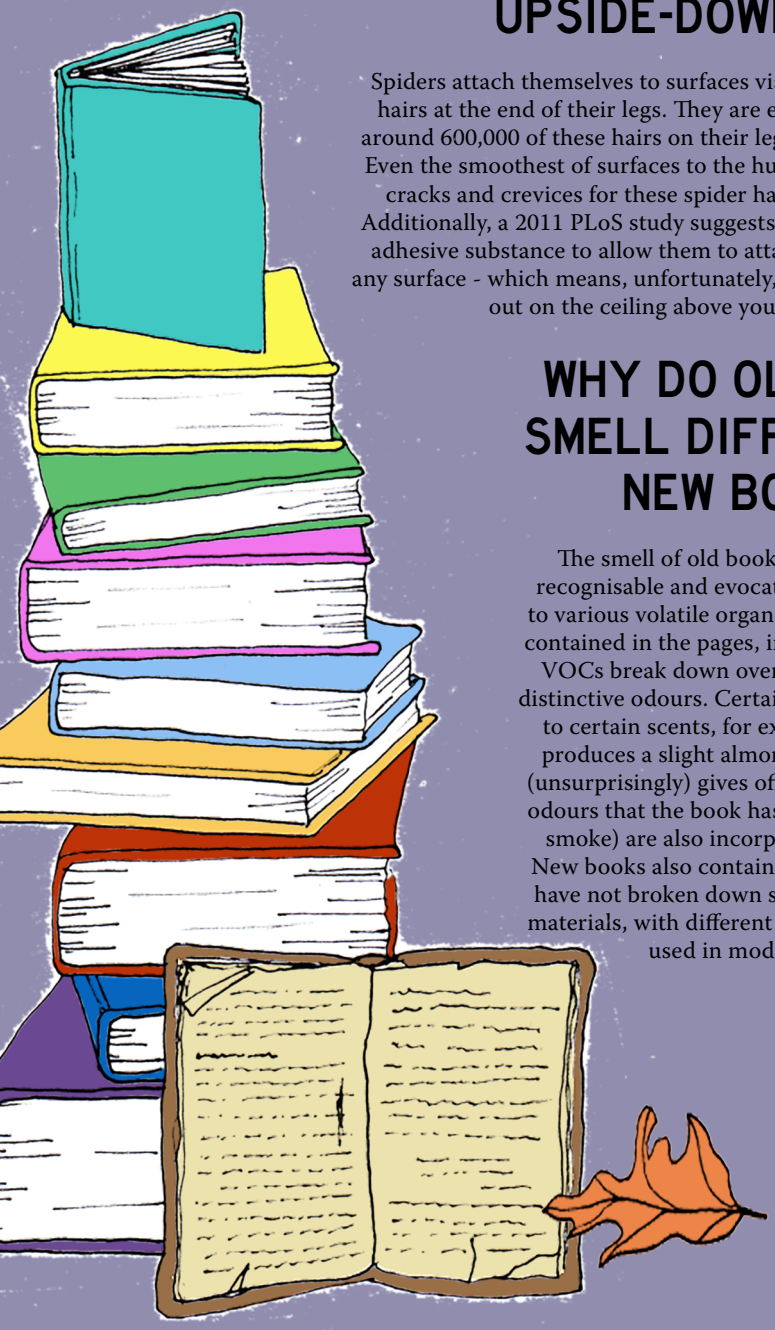
The potential implications of many species being doomed to regional or local extinction, even with no further environmental pressures, are understandably wide-ranging. Most obviously, if a debt exists, there will be a window of conservation opportunity to try and prevent it being paid. There has not been much research into the best ways to do this, as empirical examples of how and where predicted extinctions can be prevented by concentrated conservation action are rare, although there are a few examples where such action has been successful. It is also essential for conservation managers to accurately determine whether any extinction debt exists in protected areas: simply designating an area as protected can shield species within it against future threats but we now know there may be unpaid debt from historical pressures.

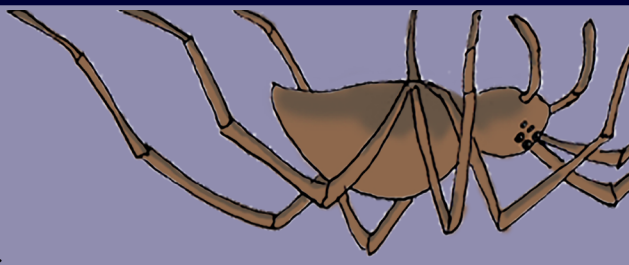
Ultimately, then, extinction debt is a frontier for biodiversity conservation. It has the potential to be incredibly important, yet we know very little about it. The fact that many species in protected areas could be declining to extinction, based on past threats that are no longer acting is definitely worrying, but our lack of knowledge means we don't really know if this is the case. Extinction debt needs continued focus if we are to save species before those windows of conservation opportunity close for good. ■

Benno Simmons is studying for an MRes in Ecology, Evolution and Conservation Research

SPEEDY SCIENCE

Editors-in-Chief, Iona Twaddell and Jennifer Toes, answer your quick-fire science questions in 150 words or less.





HOW DO SPIDERS WALK UPSIDE-DOWN?

Spiders attach themselves to surfaces via the tiny, flexible hairs at the end of their legs. They are estimated to have around 600,000 of these hairs on their legs at any one time. Even the smoothest of surfaces to the human eye will have cracks and crevices for these spider hairs to adhere to. Additionally, a 2011 PLoS study suggests spiders secrete an adhesive substance to allow them to attach themselves to any surface - which means, unfortunately, they can also hang out on the ceiling above your bed.

WHY DO OLD BOOKS SMELL DIFFERENT TO NEW BOOKS?

The smell of old books is one of the most recognisable and evocative smells, and is due to various volatile organic compounds (VOCs) contained in the pages, ink and adhesive. These VOCs break down over time, releasing their distinctive odours. Certain chemicals are related to certain scents, for example benzaldehyde produces a slight almond smell, and vanillin (unsurprisingly) gives off a vanilla scent. Other odours that the book has been exposed to (like smoke) are also incorporated into its smell. New books also contain these VOCs, but they have not broken down so much, and different materials, with different VOCs, may have been used in modern books.

WHY DO LEAVES CHANGE COLOUR IN AUTUMN?

As the days get shorter in the autumn, we see our deciduous trees turn from green to gold to red, before shedding their leaves altogether. Leaves are green because of the abundance of the pigment chlorophyll, which is important in photosynthesis. With less sunlight, there is less production of chlorophyll and it begins to break down. Slowly, other pigments, such as yellow carotenoids, can be seen - no longer masked by chlorophyll's green. As the sugars stored in the leaves break down, leaves can often turn red due to the pigment anthocynanin. Temperature can also affect leaf colour in autumn: the most vivid colours are yielded from sunny days followed by cold nights.

Illustrations by Kate Whittington

HOW DO BARCODE SCANNERS AVOID MISTAKES?

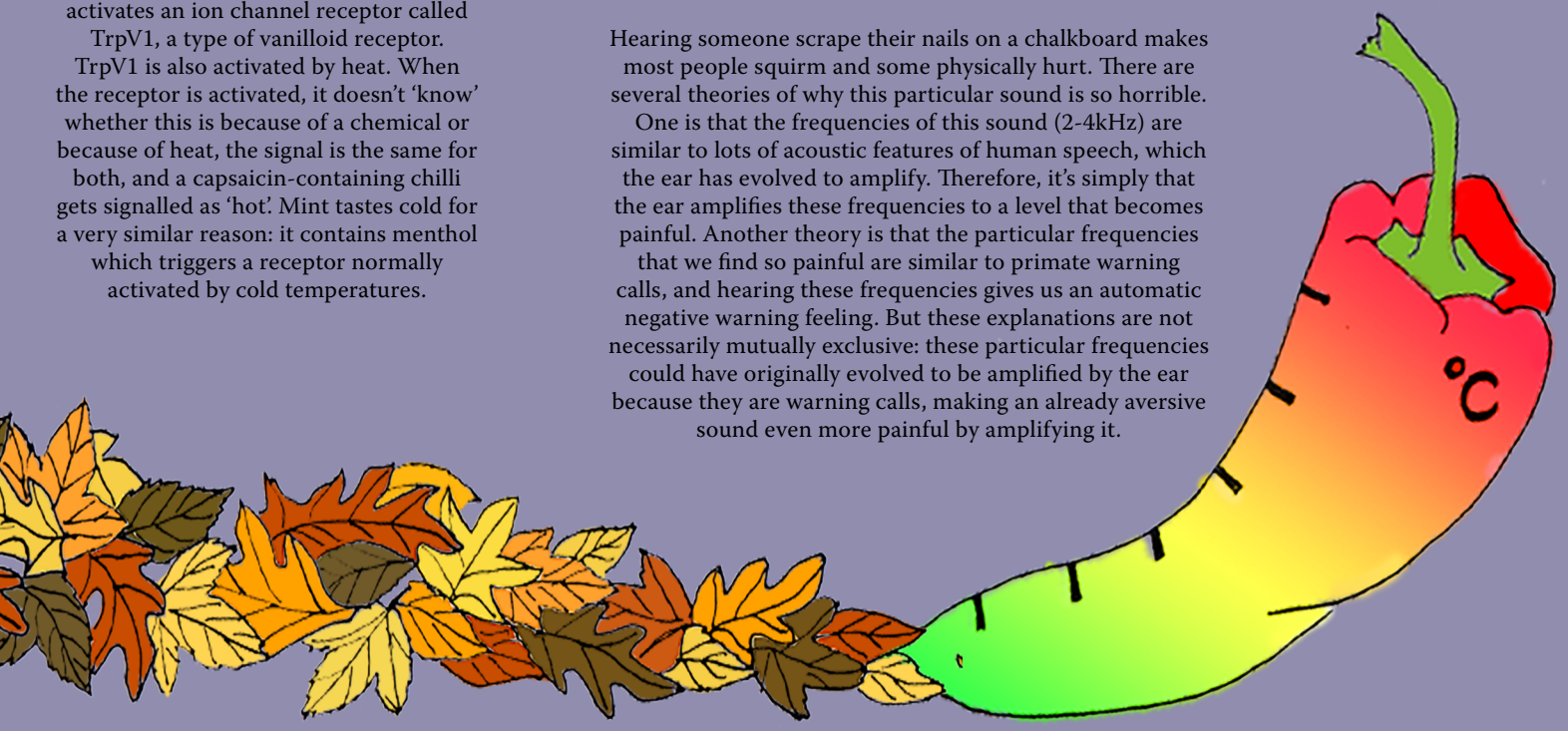
The barcodes we see on products on the shelves are made up of either a 12-digit 'Universal Product Code' or a 13-digit 'European Article Number', and the pattern of lines of varying widths above them. The code can be divided up into the manufacturer number and item number. The last digit of both types of these codes is a 'check number' to ensure the code has been scanned correctly. Barcode scanners perform a calculation of the numbers in the UPC code, and will only scan if this calculation yields a multiple of ten.

WHY DO CHILLIS TASTE HOT?

Chillis taste hot because they contain a chemical called capsaicin. Capsaicin activates an ion channel receptor called TrpV1, a type of vanilloid receptor. TrpV1 is also activated by heat. When the receptor is activated, it doesn't 'know' whether this is because of a chemical or because of heat, the signal is the same for both, and a capsaicin-containing chilli gets signalled as 'hot'. Mint tastes cold for a very similar reason: it contains menthol which triggers a receptor normally activated by cold temperatures.

WHY IS THE SOUND OF NAILS ON A CHALKBOARD SO HORRIBLE?

Hearing someone scrape their nails on a chalkboard makes most people squirm and some physically hurt. There are several theories of why this particular sound is so horrible. One is that the frequencies of this sound (2-4kHz) are similar to lots of acoustic features of human speech, which the ear has evolved to amplify. Therefore, it's simply that the ear amplifies these frequencies to a level that becomes painful. Another theory is that the particular frequencies that we find so painful are similar to primate warning calls, and hearing these frequencies gives us an automatic negative warning feeling. But these explanations are not necessarily mutually exclusive: these particular frequencies could have originally evolved to be amplified by the ear because they are warning calls, making an already aversive sound even more painful by amplifying it.



E FOR ENVIRONMENT: INTRODUCING ZERO EMISSIONS FORMULA E

Photo: WikiMedia Commons/ Patrick Ch. Apfeld

In an increasingly environmentally conscious world, the inception of Formula E, an entirely new discipline in the Formula series, seems like a solid step towards addressing climate change. Backed by the likes of Leonardo Di Caprio & Jarno Trulli this sport has all the glitz and glamour of Formula 1, but with zero emissions. For this inaugural season the FIA (the International Automobile Federation) have, for the first time, approved a car for team-wide use. Whilst all the teams are currently racing the same Spark-Renault SRT_01E provided by French-based company Spark Racing Technology, from the second season Formula E will become an 'open championship' allowing teams to create their own car from scratch. But how is this technology achieved? What makes Formula E so special, and what impacts can this have on us, outside of racing?

The technology needed to create an electric race car for Formula E is no small feat. Developing a battery that can make a car race at speeds of 150mph for one hour requires a great deal of scientific and engineering prowess as well as a large helping of creativity on the side; these racing machines are nothing short of works of art. The super lightweight chassis is made out of carbon fibre and aluminum, and meets all the safety standards of the FIA (the same standards that all Formula 1 cars have to meet). And if you thought this electric beauty is simply there to be seen but not heard then you would be mistaken: on average, the racecar produces around 80

Arutyun Arutyunyan explores the technology behind and impacts of behind the environmentally friendly racing sport

decibels at high speeds, which is more than the average road car (they sit at 70db).

The electric powertrain and electronics are supplied by McLaren Electronics Systems whilst the engineering marvel that is the battery is the brainchild of their Formula 1 rival Williams Advanced Engineering. With a cell weight of 320kg it's a hefty little thing but with its bulk comes a great deal of power. Limited, by FIA rules, to a maximum power of 200kW and an output of 28kW/h this battery produces the equivalent of 270bhp (brake horsepower) allowing it to reach a top speed of 225km/h (150mph) and do 0-100km/h (0-62mph) in three seconds. That's the same as a Lamborghini Aventador. While the Lamborghini guzzles a bucket load of petrol, the Spark-Renault simply produces a delightfully futuristic whirring sound.

Though the Williams battery is a good deal bigger than its F1 counterpart, the hardware architecture is identical. It has a built-in Faraday cage and thermal barrier to ensure the complete safety of the driver during accidents, and as we saw in the maiden Formula E Grand Prix in September when Prost and Heidfeld crashed on the final turn, accidents do happen. With an imposed restriction of 50 minutes on charging time, Williams Advanced Engineering had another hurdle they needed to overcome. How is this enormous battery, which had been racing at full capacity during qualifying, charged without overheating? This was expertly resolved by installing a liquid cooling system, a phenomenal achievement.

However, the technology still has a way to go. Drivers still need to make one stop per race to swap cars which, believe me, looks as ridiculous as it sounds.

Aside from the technology there are a lot of other things that make Formula E special. For one, they've introduced a new idea never before seen in the FIA realm – the 'Fan Boost'. This allows fans to interact with the sport on a scale previously unheard of. By voting for their favourite racer before a Grand Prix they can activate a 'Fan Boost' for the driver with the most votes, allowing the racer to add an additional 30kW (the equivalent of 40.5bhp) of power to their car for five seconds per car (drivers use two cars per race). In this modern social media obsessed world, the race organisers have really come up with a winner, getting fans more invested into the sport and adding that extra bit of excitement to each race.

> "THIS SPORT HAS ALL THE GLITZ AND GLAMOUR OF FORMULA 1, BUT WITH ZERO EMISSIONS"

Formula E has also seen, for the first time since 1976, two first-string female drivers (Katherine Legge and Michela Cerruti) in a Formula Championship. Though Formula 1 has Susie Wolff, she is still a test/reserve driver and is yet to start a Grand Prix.

It's all well and good to discuss the brilliance of Formula E in the racing world but how can its revolutionary technology help us? To answer that question we can simply look at Formula 1 and what that has done for the advancement of technology. You'll be pleasantly surprised to know that Formula 1 hasn't just made our road cars faster and safer, but has had an impact on our lives in various other ways. This includes the 'Baby Pod II', a device modelled on the driver's cockpit, allowing paramedics to safely transport newborns, to the 'Boiler Buddy' and a carbon fibre all terrain wheelchair. It's still too early in the life of Formula E to determine how or when exactly this innovative sport will transform our lives, other than significantly improving the tech behind our electric cars. But it will probably do so sooner rather than later, due to the increasing pressure from the UN for countries to cut down their greenhouse emissions. With no carbon monoxide, volatile organic compounds or lead (amongst other toxic substances) expelled by electric cars, the air will become significantly cleaner and greenhouse gas emissions will likewise see a decrease. Formula E hopes to help spark the development of our electric cars and make them more efficient by improving the storage density of their batteries as well as improving charging times and life span.

It also seems fitting that a sport so focused on 'cleaning up our cities' has all of its Grand Prix right in the middle of them. With the Beijing race circling the magnificent Bird's Nest, the host stadium during the 2008 Beijing Olympics, and the final race of the season being hosted in our very own Battersea Park, the racing looks to make a big impact on our cities. So do make sure to tune in, the London Grand Prix takes place on the 27th June 2015, and perhaps even join in on the voting for the coveted Fan Boost.■

Arutyun Arutyunyan is studying for an MSc in Advanced Materials Science and Engineering

MATHEMATICS AND EBOLA

Andrew McMahon takes a look at how we can use maths to help fight disease.

The Ebola outbreak that began in West Africa in March 2014 tapped into the public imagination across the world as representing all that can be frightening about a virus. Many commentators have spoken about its horrifying symptoms and high mortality rate, with a World Health Organization status report at the start of November declaring a total of 4,818 reported deaths from Ebola since the outbreak began.

Ebola, however, has not been the only disease to cause widespread fear this century. After the SARS outbreak of 2002-2004 and the swine flu pandemic of 2009, modern history has taught us that viruses can spread very quickly throughout local populations and even across the world. These recent examples left many ordinary people asking: how do we understand when an outbreak will become a pandemic? When is it best to start investing in mass vaccination or producing large amounts of combative drugs? How can we predict how severe an outbreak will be?

The answers to these questions can be found, in part, using mathematics.

By applying the tools of mathematics to epidemics, scientists and public health officials can make more informed decisions about the best course of action to take. For example, if a mathematical model can accurately describe what effect mass vaccination will have on an outbreak, a decision can be made about whether this is the most effective way to spend (ultimately finite) resources. Minimising the risk associated with these types of choices is one of the main goals of the mathematical study of epidemics.

It is clear that having a predictive model of a disease would be useful, but how does this work in practice? To write a mathematical model of an infection, you first need to identify what the important variables are: how contagious is the disease? How long does it take before an infected person becomes infectious to others? Will the rate of infection change with time?

In fact, almost all basic models of epidemics will contain variables describing the answers to the above three questions. Respectively these are called the reproductive number, the generation time and the epidemic growth rate. These numbers give you the basic information about any epidemic, but in order to capture the correct behaviour, more detailed models take into account other factors such as demographics, migration, transport networks, seasonality, or even sexuality and drug use (as in the case of AIDS).

Using the above numbers you can write down an equation that should tell you how many individuals in a population are infected at any one time. If your model is good enough, you can then use it to predict the effect of introducing new variables into the system, for example the effect of having a mass vaccination program. This sort of information can prove invaluable to public health officials as it allows for the analysis of several possible scenarios without wasting the precious resources of time and money testing them in the real world, ultimately saving lives.

Once a model is developed, the predictions can be somewhat surprising. For example, some models of the H1N1 virus (swine flu) produced during the 2009 pandemic predicted that if developed world countries focussed on vaccinating and treating only their own population, a serious global pandemic could still not be avoided. The interconnected world of the 21st century meant that resources would have to be shared between developed and developing countries in order to beat the virus. In the end however, it turned out that H1N1 was a mild strain with a relatively small mortality rate, so the pandemic was not as serious as predicted. The results from this model could, however, still prove useful if and when a serious pandemic hits, as many experts believe could occur with a future strain of avian flu.

There are limitations in terms of what mathematics can offer, however. Many patterns of infection require very sophisticated models in order to understand them, with many not being successfully modelled yet at all. The fact that mathematical epidemiology is such a vibrant area of research is a testament to the fact that there is always more to do. That said, in the age of 'Big Data' and vast computing resources, epidemiological models are getting better all the time. This will hopefully mean that when the next potential pandemic hits, we can be confident of containing it. ■

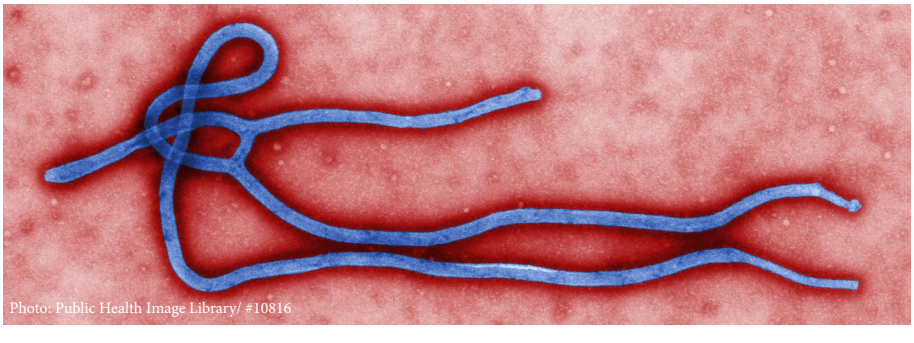


Photo: Public Health Image Library/ #10816

Andrew McMahon is a first year PhD student in Physics

UNFOLDING THE TRUTH ABOUT PRIONS

Rapidly multiplying misfolded proteins undetected by the human immune system - Sophia Ho explains why prions are important in understanding diseases.

Creutzfeldt-Jakob Diease, Kuru, Fatal Familial Insomnia - in what may seem like a list of ruthless and incurable neurodegenerative diseases lies a littler known, yet remarkable phenomenon: the prion.

Prions are not bacteria, viruses, parasites or even fungi. They don't result from malfunctioning immune systems. Yet they are extraordinary as they can be propagated through three very different modes of disease transmission; they can be inherited, spread via contaminated materials, or simply occur spontaneously.

But what is this mysteriously unstoppable prion, which can cause convulsions, behavioural changes, loss of coordination and dementia, often followed by fatality within months of onset?

The prion, whose name comes from a bizarre combination of the words 'protein' and 'infection', is merely the misfolded form of a normal protein known as PrPC, which is found throughout the body and is responsible for attachment and cell signalling. Proteins exist in unique shapes which are essential for their function, therefore the loss of this shape can make it lose its function, or even become toxic to the cell. This forms the basis of many disorders, including those listed above and others such as Alzheimer's and Huntington's disease.

Prions, however, differ hugely from other misfolded proteins in their ability to self-propagate and multiply. As prions are not living structures, they are often compared with viruses. However, this assumption is incorrect, as unlike viruses, genetic material is not necessary for their function or spread (excluding the inherited forms causing 10-15% of all prion disease).

How then, does a prion come about? How can it spread if there's no genetic material to replicate? Even now scientists can only hypothesise the processes underlying prion diseases.

For hereditary forms at least, such as Fatal Familial Insomnia, it has been established that mutations in the PRNP gene lead to prion proteins that are more likely to fold abnormally. For sporadic and transmissible forms, the cause is still a mystery. However, it is known that the shape of any protein can be affected by changes in its environment, such as temperature or acidity, causing the unfolding of a protein to an unstable form.

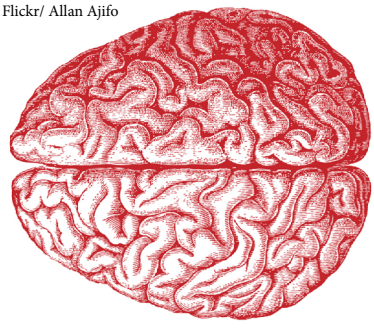
The unstable form of prion, known as PrPSc, tends to clump, or aggregate, with regular PrPC proteins. In doing so, it causes these to convert to PrPSc at an exponential rate, leading to a fatal chain reaction.

Studies have suggested that the immune system not only tolerates this dangerous difference, but may even unknowingly aid their spread throughout the nervous system and brain, where aggregation fatally occurs.

This aggregation destroys tissue and creates plaques that eventually form holes in the brain itself, leaving it a 'sponge-like' structure. It may be appropriate then that prion diseases are known as spongiform encephalopathies (SEs).

SEs are famously associated with cannibalism practised by tribes in Papua New Guinea, leading to Kuru, and the feeding of cattle meat to other cattle, which led to

Flickr/ Allan Ajifo



the outbreak of Creutzfeldt-Jakob Disease (CJD) in the UK in 1986. This resulted in the deaths of 166 people, and the infection and culling of 4.4 million cattle. In 2013, it was estimated that 1 in 2,000 people in the UK still carry the CJD-causing prion (which may continue conversion without symptoms for decades).

CJD is of continual global concern. As recently as September 2013, Russia finally lifted its 26-year ban on British beef imports. Many countries also ban blood donations from individuals who have lived for at least three months in the UK between 1980 and 1996, or at least five years in any European countries from 1980 until now.

Promising diagnostic and therapeutic measures are continually being developed to detect and prevent PrPSc accumulation. Nonetheless, it is astonishing that the cause behind such harrowing and terrifying disorders is a simple error, with no seeming cause at all. ■

Sophia Ho is studying for an MSc in Molecular Medcine

THE GREAT DEBATE: SUGAR VS. ARTIFICIAL SWEETENERS

Suger is one of the oldest cooking ingredients, dating back to 326 BC. Since then it has been successfully used for many different purposes: in healing wounds, growing penicillin, leather tanning, printer's inks, dyes and even to prolong the life of freshly cut flowers. But how beneficial is the consumption of suger for human health?

Simple sugars are composed of monosaccharides (glucose, fructose and galactose), while granulated sugar, most commonly used in food, is composed of sucrose – which is broken down to glucose and fructose once it enters the body. In the last few decades, the food industry has started producing synthetic food additives that mimic the sweet taste of sugar: now widely known as artificial sweeteners. At the same time, granulated sugar has been characterised by both scientists and nutritionists as a harmful and dangerous ingredient that should be avoided, while they have encouraged the consumption of artificial sweeteners. But to what extent is this perspective true?

Both sugar and artificial sweeteners are composed of glucose and fructose, though fructose levels are ~5% higher in the majority of artificial sweeteners. Since fructose does not have any significant impact on leptin (the hormone responsible for regulating the sensation of hunger), you may still feel hungry after consuming products containing artificial sweeteners compared with products containing sugar. This could lead to the over-consumption of food containing artificial sweeteners which can result in obesity.

For many decades, scientists and dentists have supported the statement that the consumption of sugar increases the risk of tooth decay, whereas the consumption of artificial sweeteners does not have any negative impact on oral health. However, new studies from Johns Hopkins University have demonstrated that the regular consumption of artificial sweeteners can cause dental erosion.

Glycolytic acid, found in sugar, is very beneficial for the maintenance of healthy skin. Due to its hydrating effect, it can diminish blemishes and treat sun-damaged and aged skin. More specifically, skin products containing simple sugars draw moisture from the environment which nourishes, hydrates and restores the balance of oil in the skin.

Sugar is also very beneficial during exercise. The consumption of carbohydrates in the form of simple sugars during exercise increases the durability of performance. This is because sugars are quickly absorbed in the gut, transported into the muscle and transformed into energy. In contrast, artificial sweeteners are composed of long acting and complex carbohydrates which decrease endurance; they are absorbed gradually and first must be transformed into sugars before they can be transported into muscles and metabolised into energy.

Sugar contains elements that are beneficial to our health, including magnesium, potassium, iron, calcium and phosphorous, which we can easily metabolise. In contrast, artificial sweeteners are industrially processed and do not contain great quantities of natural elements. This can cause digestion problems. Additionally, most artificial sweeteners contain aspartame which causes headaches after long-term consumption. There is also evidence that the long- term consumption of artificial sweeteners can cause bladder cancer in laboratory animals; however the outcome from subsequent carcinogenicity studies of artificial sweeteners has not provided strong indication of a link with bladder cancer in humans. Accordingly, sugar consumption is both beneficial and essential for the maintenance of our health. As usual though, it's best to take 'everything in moderation', as excessive sugar intake usually results in severe health problems. ■

Angelina Chrysanthou is studying for an MSc in Molecular Medicine

Stephanie Sammann and Angelina Chrysanthou battle it out over which of these everyday foods really presents the greatest health risk.

Illustration by Pui Sham

The debate over artificial sweeteners and cane sugar seems endless. Some say that artificial sweeteners are worse than real sugar because they are "just chemicals". Others seem to understand the enormous amount of calories in even the smallest amount of sugar. Some say artificial sweeteners cause cancer, while others say it is cane sugar that in the end gives you cancer. Is there any weight to these claims? Is one option measurably healthier and safer than the other?

The answer is, yes, most likely. Despite the numerous assertions in both directions, scientists, nutritionists, and doctors have seen the evidence that refined sugar is far more likely to cause long-term health problems than artificial sweeteners. In a world where more than 35% adults are overweight, and 350 million people suffer from diabetes, artificial sweeteners are clearly something to consider.

According to sugar and diet researcher David Gillespie, the average Briton consumes more than a kilo of sugar a week. This comes from products containing corn syrup, agave or maple syrup and honey. The food industry loves these sweeteners, especially high fructose corn syrup because it makes every type of food more palatable – from soup to cereals, ketchup to bread. What we see as every day essentials are pushing us over the healthy limit for sugar intake. This is worrying when obesity rates are continuously rising.

Currently 26% of Britons are obese, and half are overweight. This problem extends to more than just personal health. The direct costs caused by obesity are now estimated to be £5.1billion per year. Obesity is associated with cardiovascular risk, cancer, disability during old age, decreased life expectancy and serious chronic conditions such as type 2 diabetes, osteoarthritis and hypertension. This puts a big strain on the economy, and on the happiness and livelihood of the population.

Because our sweet tooth seems insatiable, it seems implausible that everyone will realize the cost of sweetness and erase it from their diet. Instead, it may be time to put the myths about the dangers of artificial sweeteners to rest and incorporate them more heavily into the national diet, giving people their sweet fix while keeping some of those ill effects at bay. While it is essential to consume artificial sweeteners in moderation, just like anything else, they could help many people cut out sugar from their diets.

There are three main types of sugar substitutes: artificial sweeteners such as aspartame, sugar alcohols such as sorbitol, and novel sweeteners, which are derived from stevia. Each of these has low or no calories, do not raise blood-sugar levels, and are safe for diabetics to consume.

If you are worried about the health risks of sweeteners listen to this: the idea that sugar substitutes are carcinogenic first surfaced in the 1970s when saccharin (found in Sweet'N Low) was discovered in one study to raise the risk of bladder cancer in rats.

A wealth of later research in humans has found no link. Similarly, aspartame, the most commonly used sweetener, was blamed in 1996 as the cause of the spike in brain tumours in Americans between 1975 and 1992. Again, further studies have found no connection. Many scientists and nutritionists say we should take the claims of the cancer causing properties of sweeteners with a grain of salt.

While switching to these sugar-free products should be paired with a low-fat diet and regular exercise, it can be a way to kick-start a healthy lifestyle. ■

Stephanie Sammann is studying for an MSc in Science Media Production

COELIAC'S DISEASE

Sophia Ho reveals the real impacts of gluten intolerance

It was in 2007 when Karen started to see the signs. Working in the police force for over 20 years, she was an exceptionally healthy and athletic individual.

Seven years ago, however, some changes became apparent. Karen began to frequently feel tired, lethargic, out of breath with her pulse racing. She also felt bloated after virtually every meal, lost weight and even noticed some thinning of her hair and eyebrows.

As Karen had family members that suffered from hypothyroidism, an underactive thyroid, she asked her doctors to test her for the same condition. It turned out that Karen suffered instead from hyperthyroidism, an overactive thyroid, for which she was given medication.

However, after taking the medication, which restored her normal thyroid activity, Karen still experienced the same symptoms as before. In addition, she continued to gain weight and no amount of exercise allowed her to lose it. Most surprisingly, she started to develop vitiligo (partial skin depigmentation).

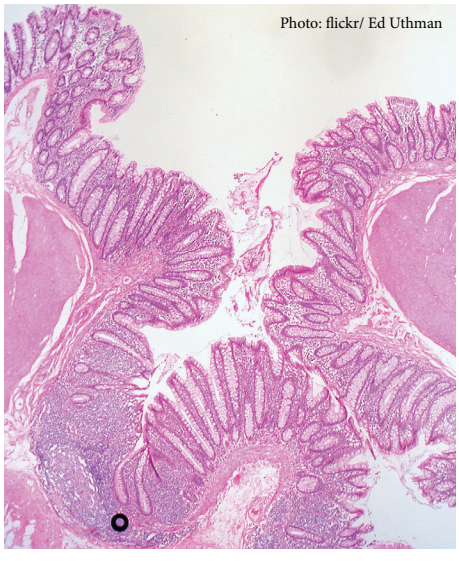


Photo: flickr/ Ed Uthman

The issue finally resolved when Karen decided to seek a private consultant and endocrinologist, who at long last was able to diagnose her as a sufferer of Coeliac's disease, confirmed via a gut biopsy. Previous blood tests had not been able to establish this, which is not uncommon for Coeliac patients.

Coeliac's disease (CD) is an autoimmune disease that affects the large intestine in response to a protein found in wheat, barley and rye, known as gluten. Gluten gives dough elasticity and allows it to rise, and can be found in various food products such as bread, pasta, chocolate and soy sauce. Coeliac's disease has been documented throughout history, from as early as the first century AD, as a condition that manifests as malabsorption, diarrhoea or constipation, weight loss, bloatedness, fatigue and stomach pains.

Upon exposure to gluten, an intestinal enzyme known as tissue transglutaminase modifies its soluble component, gliadin, forming a new complex. In Coeliac patients, this new complex triggers an inflammatory response, which destroys epithelial cells in the small projections lining the intestine, known as villi. This leads to the impaired absorption of nutrients in the intestine, and to the symptoms listed above and those Karen experienced. Indeed, the biopsy taken from her revealed that her villi had largely deteriorated.

The only known and successful way to deal with CD is to exclude gluten in your diet for life.

Current research has focused on generating genetically modified wheat designed to reduce stimulation of the immune response. Alternative solutions have looked at ways to produce ingestible enzymes that remove the stimulatory gliadin protein and prevent it from triggering inflammation.

CD is fairly common and can even be hereditary - it is believed to affect up to 1% of the world's population. Many remain undiagnosed, as symptoms may not always be presented, or, as in Karen's case, don't manifest until adult age.

After seeing the consultant and following his recommended diet, Karen's condition dramatically improved: her hair started to regrow, and her energy levels and weight returned to normal. Further tests on her hyperthyroidism also proved that it was caused and influenced by her issues with CD, and most likely won't affect her again provided she continues to avoid gluten-containing foods. On the other hand, her vitiligo, also caused by CD, may stay with her for life. However, a biopsy taken a year ago revealed that the villi lining of Karen's intestine was restored to normal.

Only one small concern remains. We mentioned that CD is known to be a genetic predisposition. Karen's consultant has stated that as many as 85% of her family may also be 'Coeliacs'. It may now be time to reveal that Karen is in fact my maternal aunt!

Karen's condition now hardly affects her; her life has returned to normal and we always ensure that her needs are met at every family food gathering. In the meantime, we shall all be on the lookout for any symptoms of our own! ■

Sophia Ho is studying for an MSc in Molecular Medicine

REVIEWS

BOOK

by Bentley Crudgington

THE DELUSIONS OF GENDER: THE REAL SCIENCE BEHIND SEX DIFFERENCES

BY CORDILIA FINE

You may have heard that gender inequalities are a natural part of the human condition. Once you understand the science you too will agree. In the darkness of the womb our brains are soaked in a cocktail of sex hormones which 'hardwire' gender into our neurons. All proceeding actions are just a consequence of this, it's pre-defined, it's science! But as Cordelia Fine says in her introduction "If only it was that easy" and just like gender, this book is complicated.

Delusions of Gender does not read like a popular science book but more an impeccably researched thesis with manifesto tendencies. The overall feel is more of a meta-analysis and the dense referencing can make some sections rather fragmented. The moments of sardonic humour are more from eye rolling at the ridiculous and out-dated theories on gender rather than from the writing itself.

Where this book excels is in the author's insightful and critical thinking. Research methods, definitions and context are all unpicked to show how a gender bias is introduced before the 'science' has even happened.

One example is 3D rotation, the ability to identify a matching pair within a panel of similar rotated objects. In the standard test, 75% of those scoring above average are male. If participants are told they are being tested for their engineering skills then this percentage increases. However if they are told that it was to assess interior design or needlework skills, then on average females outperform males on the test: the 'real science' behind sex differences disappears.

Systematically showing experimental flaws and challenging assumptions made in the research is great but repeatedly annihilating the same assumption serves little purpose. If Fine discussed fewer examples in more depth, maybe they would stick longer in the mind and make it into everyday conversations.

This book does have some biases of its own which is understandable given that the impact of gender inequalities is more detrimental to women than men. However the words sex and gender are used almost interchangeably and most examples are heteronormative.

After reading this book, you will certainly become more aware of both conscious and unconscious gender biases and how manipulating and insidious gender stereotypes can be, which can only a good thing - you will just have to work for it. ■

Bentley Crudgington is a second year PhD student studying Virology

EVENT

by Charlotte Mykura

RULES OF ATTRACTION: LATES AT THE ROYAL INSTITUTION

For the first time in history, this 24th October 2014 the Royal Institution (Ri) opened its doors for an adults-only lates event, with the theme Rules of Attraction.

Situated in Mayfair, the beautiful Ri building has been home to some of the world's greatest scientists since 1799. Inside, the building boasts sweeping staircases, gorgeously old bookcases, the circular lecture theatre (famously known from the Christmas lectures), and a downstairs laboratory.

Entering the Ri, I was immediately taken by the sound of live jazz and friendly chatter. Gleeful guests wandered and pondered, learning about sexual natural history whilst sipping on wine or eating chocolate-dipped marshmallows. Many visitors exhibited wild, colourful headdresses that they had made themselves at the upstairs Birds of Paradise plumage table.

A feast of knowledge was available at the different stands, from taking part in a heart dissection to learning about the evolution of sexual characteristics. I could hold stick insects and gaze at their sexual dimorphism, dissect a flower, play with Ferrofluid and magnets, or explore the latest facial recognition software. Never before have I been at such an interactive and engaging scientific event.

As well as enabling you to explore your creative side and get your teeth into some serious science, this first ever lates event was a great night out. The crowed spanned all adult age groups, who enjoyed the blues dance class together, had fascinating conversations and learnt collaboratively about all topics sexual.

Several short lectures took place in the Ri theatre during the course of the evening. Guests learnt about the courtship routines of different species, including the sexy erectile snood of male turkeys and the guppy fish's fiery colours. In the talk entitled 'Secrets of Animal Sex', some extremely strange sex lives were unveiled.

The evening had a great vibe; it felt almost like a ball – but sciency. The night was perfectly pitched (I didn't queue once, always had something fun to do) and I've come away from 'Rules of Attraction' with a head full of weird and wonderful facts.

A quirky, energetic, classy, creative, science night out. Definitely a night that I'll remember. ■

Charlotte Mykura is a second year PhD student studying Chromatin Biology